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SUBCORTICAL HEMORRHAGIC CYST BENEATH THE ARM AND LEG AREAS.[1]

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THE following case is reported chiefly with a view of emphasizing a single point in the differential diagnosis of a limited lesion of the motor sub-cortex.

R. J. P., sixty-six, white, a seaman, had an attack of subacute rheumatism twenty-two years before coming to the hospital. No history of syphilis or alcoholism could be elicited. On a cold day in January, 1888, while in a wagon, he had a "queer feeling," and found that he had lost power in the right leg and arm. He did not fall or become unconscious. He at first could neither sit or stand, and has never since been able to stand.

In February, 1888, about one month after the paralysis, suddenly and without warning, his right hand and forearm began to twitch. He was at first entirely conscious and watched the progress of the seizure. The arm was first drawn upward, then the fingers and hand were flexed, next the forearm was bent upon the arm; then the arm—from his description, spastic—was elevated. Next his head and neck began to twist to the right, his right face also twitching. He thinks he became unconscious. This is a description of the seizure as he was able to observe it five times since February, at intervals of about a month apart.

He had a sixth and last fit about the middle of July, 1888. In this, after the arm, face and head had been affected in the usual serial order, according to the above description, the spasm passed into the right leg before he became unconscious. He could not give as accurate an account of the leg spasm as of that affecting the arm and face, but, from his statement, the leg probably flexed on the thigh and became very rigid with shaking movements.

He had no vertigo, no double vision, no tinnitus, no nausea, no headache, and no pain in the arm, face, nor leg, except sometimes a cramp-like feeling. Shortly after the first spasm, the right foot and leg swelled, and it had since

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done this frequently. He had some tremor of the tongue. Faradic and galvanic contractility were retained.

The knee-jerk on the left side was about normal; no muscle-jerk or ankle clonus. On the right side knee-jerk and muscle-jerk were much exaggerated, and ankle clonus marked; no wrist-jerk, elbow-jerk, nor jaw-jerk could be elicited. Touch, pain and temperature senses were normal, as were also taste and smell. The pulse usually ran 85 to 95 per minute. The arteries were markedly atheromatous. The heart sounds were regular and forcible, the sound at the apex accentuated, but no murmurs could be heard anywhere.

The most persistent and perhaps the most distinctive symptom in this case, was a continuous spasticity of both arm and leg, but particularly the latter, on the paretic side. The right leg remained all the time in an almost rigid condition, slightly flexed at the knee and in a state very similar to that of both limbs in many cases of spasmodic tabes; it was indeed an almost typical spastic paralysis of this extremity. The right arm was also markedly spastic, but the rigidity was not so extreme as in the leg.

Observations in cranial temperature were not made. Percussion and strong localized pressure were used on various occasions, but gave no indication. The patient never complained of localized pain.

One day he had a sudden and severe apoplexy, with profound unconsciousness, Cheyne-Stokes breathing, and the train of sypmptoms belonging to extensive intra-cerebral hemorrhage; but for the purposes of this report it is not necessary to go into the details of this attack which terminated fatally in a few hours.

The autopsy revealed in the right hemisphere an immense recent hemorrhage into the ganglia, tracts and surrounding parts, breaking also into the lateral ventricle—a major apoplexy in a location, and of a character often described, which was undoubtedly the cause of his death; but my chief interest was in the examination of the other hemisphere.

External examination of this left hemisphere revealed nothing but a dark spot about one-fifth of an inch in diam-

eter at the bottom of the upper extremity of the central fissure. On exploring the left lateral ventricle, a yellowish black area, one-third of an inch in length was noticed in the white substance composing the curved roof of the ventricle, about the middle of its antero-posterior extent. Carefully cutting into this it was found to be a hemorrhagic cyst, which was in the subcortex related to the middle and upper thirds of the anterior central convolution. An arm of this cyst extended to the spot of degeneration in the fissure of Rolando.

The differential point which would seem to be taught by this case is one which has already been alluded to by Lloyd and myself, as well as by Seguin and others—that is, the predominance and persistence of tonic spasm in cases of subcortical lesion. Numerous as have been the reports on cases of restricted and of diffused motor cortical lesions, few instructive cases of isolated subcortical lesion have been recorded, and these have been mostly tumors. Efforts have been made to designate the localizing signs and symptoms of such growths by Seguin and Weir, also by others, as Osler, Lloyd and the writer, Bernhardt, Nothnagel, and Jackson. Franck made numerous experiments on the cerebral motor fasciculi, the most important facts demonstrated by him being that electrical excitation of these fasciculi produces only tetanic contractions, ceasing with the excitation. The one fact which would seem to be emphasized by the record of this case is that a lesion partly destructive and partly irritative—such as was the local hemorrhage which subsequently degenerated into the cyst revealed by this autopsy—has for its most characteristic symptom a persisting spastic paresis or paralysis. The attacks of beautifully characteristic Jacksonian epilepsy which the patient experienced, were probably due to cortical—not subcortical—irritation,as the lesion at one point slightly invaded, from within outwards, the cortex; and even if this invasion had not taken place, as Seguin has suggested, irritation may be radiated centrifugally as well as centripetally from subcortical lesions to the neighboring gray matter.

In another case, with autopsy, a report of which will be made at the present meeting, the lesions which were bilateral, were also both cortical and subcortical, and partly in the motor subcortex; and one of the most striking of several striking phenomena was persistent myotonia.